DOCUMENT RESUME

ED 182 884 EC 122 924

AUTHOR Schofield, Leon J.
TITLE Hyperkinesis: Recent Diagnostic and Treatment Trends.
PUB DATE Oct 79
NOTE 22p.: Paper presented at the Annual Genesee Valley Psychological Association Conference (Rochester, NY, October 19, 1979)

EDRS PRICE MF01/PC01 Plus Postage.
DESCRIPTORS *Clinical Diagnosis: Dietetics: Drug Therapy: Early Childhood Education: Elementary Secondary Education: *Etiology: *Hyperactivity: Psychotherapy: *Student Characteristics: *Therapy

ABSTRACT

The paper describes hyperkinesis, a common behavior problem of childhood which is characterized by symptoms which include involuntary and consistent overactivity usually with onset in early childhood or infancy, short attention span, and poor school performance. Sections touch on the following aspects: symptoms, etiology, course of illness, identification, treatment/medications (including use of stimulants), treatment/diet (such as the Feingold diet), and treatment/psychotherapy. The author concludes that clinicians need to maintain a broad, flexible attitude since various treatment approaches are effective with various individuals and since responsiveness to a specific treatment may also change over time, requiring a shift in treatment. (SBH)

****
* Reproductions supplied by EDRS are the best that can be made *
* from the original document. *
****

ED182884

U.S. DEPARTMENT OF HEALTH,
EDUCATION & WELFARE
NATIONAL INSTITUTE OF
EDUCATION

THIS DOCUMENT HAS BEEN REPRODUCED EXACTLY AS RECEIVED FROM THE PERSON OR ORGANIZATION ORIGINATING IT. POINTS OF VIEW OR OPINIONS STATED DO NOT NECESSARILY REPRESENT OFFICIAL NATIONAL INSTITUTE OF EDUCATION POSITION OR POLICY.

HYPERKINESIS : RECENT DIAGNOSTIC AND TREATMENT TRENDS

Leon J. Schofield, Ph.D.
Clifton Springs Hospital and Clinic

Presented at the annual
Genesee Valley Psychological Association Conference,
Rochester, New York, October 19, 1979

"PERMISSION TO REPRODUCE THIS MATERIAL HAS BEEN GRANTED BY

Leon J. Schofield

TO THE EDUCATIONAL RESOURCES INFORMATION CENTER (ERIC)."

EC122924

Hyperkinesis :
Recent Diagnostic and Treatment Trends

Leon J. Schofield, Ph.D.
Clifton Springs Hospital and Clinic

Introduction:

Hyperkinesis is the single most common behavior disorder seen by mental health professionals, with estimates of incidence ranging from 2 to 10 percent of the elementary school population (Ross and Ross, 1976). Males typically outnumber females, with the literature reporting ratios ranging from 3:1 to 9:1. The National Health Institute estimated that in 1971 there were over 300,000 children taking stimulant medication (Feingold, 1974). There can be no doubt that hyperkinesis is a serious disorder affecting great numbers of children.

The Syndrome and the Symptoms:

Many terms have been used to describe the syndrome discussed within this paper. Although the terms hyperactivity and hyperkinesis are currently in vogue, the terms minimal brain damage and minimal brain dysfunction have been common in the past and in fact appear to be making a comeback in the recent literature.

There is a constellation of symptoms readily identifiable and hyperkinetic individuals generally show most of these symptoms in their past history or current behavior. Although some controversy continues about when to diagnosis "hyperkinesis" (Klein and Gittelman-Klein, 1975) the term persists. This fact is a testimony to both its heuristic value and our neurological ignorance currently.

The symptoms of hyperkinesis have gradually become more refined as

clinicians and researchers have worked with hyperkinetic children. They will be noted in detail below.

Laufer and Denhoff (1957) provided one of the earliest and most comprehensive definations of hyperkinesis. The symptoms included:

1. involuntary and consistent overactivity, usually with onset in early childhood or infancy
2. short attention span
3. impulsivity, inability to delay gratification
4. anhedonia - tense, demanding, persistently unhappy, inability to experience pleasure
5. perceptual/cognitive dysfunction

To this initial outline, Wender (1971) added:

6. refractoriness to discipline measures
7. extroversion

Rapoport and Quinn (1975) observed a high incidence of:

8. anatomic anomolies (e.g. high palate, assymmetry, etc.)
9. early fussiness
10. early history of headbanging

Feingold (1974) added:

11. clumsy, poorly coordinated
12. poor school performance, despite average or above average IQ

Connors (1975) notes:

13. "driven" quality to behavior, episodic rigidity, compulsiveness
14. destructiveness of property
15. aggression with peers

16. low frustration tolerance
17. early and quick awakening

I would add to the list, based on my own clinical observations of hyperkinetic children:

18. emotional lability
19. poor response to permissiveness
20. depression, low self confidence
21. easily "set off" by others' emotion
22. tactile defensiveness (avoids being touched, held, etc.)
23. great appetite, often compulsive eating, usually without weight gain
24. rather fearless, takes physical risks, ignores pain

Etilogy:

The exact cause of hyperkinesis is unknown, but it is thought to include organic, genetic, psychogenic and environmental factors (Safan and Allen, 1975; Ross and Ross, 1976).

The presence of some unspecified underlying neurological impairment (Gittelman-Klein and Klein, 1975) is generally accepted. Hyperactive children have had greater frequencies of abnormal autonomic, EEG and evoked cortical responses, suggesting low CNS arousal (Satterfield and Cantwell, 1975). The most accepted therapy currently is that hyperkinetic children have insufficient cortical inhibitory control over both sensory input and motor functions. Stimulants, acting on the midbrain reticular activating system, produce increased cortical inhibitory control. Meeks (1975) has noted that studies have shown low CNS aroused hyperactives appear to be most responsive to stimulant medication. High CNS aroused hyperactives show more individual and family psychopathology. These differences remind us that hyperactivity

can be defensive in character in response to family stress, etc.

Etiology of "hyperactivity" might also include poor schooling, inadequate stimulation and poor nutrition in deprived subgroups (Gittelman-Klein, 1975).

Meeks (1975) proposes that we differentiate between the: (1) organically hyperactive, with more profound and obvious organicity, related to specific damaging experiences (e.g. damage during pregnancy or delivery; tumor or other lesions of the brain; infectious illness of CNS; neonatal anorexia; degenerative diseases of CNS); (2) environmentally hyperactive,with stressful and damaging social environments, related most probably to psychological factors; and (3) maturationally hyperactive child, with neurological impairment of a minor degree, producing the symptoms noted above. The present paper focuses almost exclusively on the last category.

Because the treatment strategies might vary widely amoung these subtypes, accurate diagnostic assessment is essential.

Course of illness:

The onset of hyperkinesis is generally in the preschool age child. Although some hyperactive children show signs of hyperkinesis in infancy, I have not seen. a large number of hyperactive cases where extremely excessive behavior was evident in infancy. More subtle signs may be present. It is most common, however, for parents to notice a hyperactivity problem in early childhood when the child is rapidly expanding his experiences due to greater mobility and language development. Thses children are often not referred to clinicians, peditricians, etc. until they have entered school and become still more noticably different and unable to cope with still greater demands.

Pre-school children have important learning experiences they must encounter. The limited attention, low frustration tolerance, etc. makes it difficult for the child to develop mature motor skills, social skills, problem solving skills, etc. Even at these early stages, the hyperkinetic child is falling rapidly behind his peers. Because of this and because the hyperactive young child is also so often in conflict with his environment, self confidence begins to crumble and negative self labelling occurs (e.g. "I'm dumb," "I'm a pest," etc.).

The latency years are not a time for quiescence. The child is now falling behind in school, struggling to barely pass each year. The child is often unable to participate easily with peers in sports activities because of general awkwardness and poor coordination. Self esteem diminishes still further. Manual skills are also not refined. Activity level remains high. The child is more mobile, he or she is "wearing out" others, teachers, neighbors, etc.

In adolescence, the child typically becomes less overtly hyperactive. However, concentration and attention span are often still poor and impulsivity and hostility remain. Anti-social behaviors often emerge. The normal adolescent turmoil is even more pronounced for the hyperactive child. The struggle for independence and autonomy is acute. With little self confidence and a general lack of social skills, academic skills, etc. the child frequently gravitates towards fringe sub-groups composed of other social "misfits." They often leave school prematurely. Depression is common, often masking acting out episodes.

Satterfield and Cantwell (1975) have found that about 80% of hyperactive children in early adolescence showed mild anti-social behavior (e.g. lying, impulsivity), about 50% moderate antisocial behavior (e.g. fighting, stealing)

and about 20% serious anti-social behavior (e.g. running away, truancy, excessive drinking).

While once it was felt that hyperactivity is strictly a childhood disorder, it is becoming generally more accepted that it is not just a childhood disorder.

Guze, McKinney and Cantwell (1967) studies parents of hyperactive children and found a higher prevelence of parental alcoholism, sociopathy and hysteria than among parents of non-hyperactive controls. They also found a greater percentage of the parents of hyperactive children were themselves hyperactive as children; of these parents, 84% had developed sociopathy, hysteria, or alcohol problems.

In my own clinical experience working with many alcoholics in the hospital's rehabilitation unit, I have found several instances of apparent hyperactivity in adults. The excessive activity is curbed from childhood, but the alcoholic remains inattentive, restless, impatient, often hostile. Alcohol is used as a calming drug to aid sleep, etc.

Bellak (1979) has recently edited a volume in support of the concept of adult hyperactivity. Feingold (1974) has reported examples of adult hyperactivity as well. Considerable research is needed, and this appears to be a promising area for further attention.

Screening for hyperkinesis:

For children who are blatently excessively active the diagnostic process is easy. When we see such children in our offices, we spend most of the time following them around trying to prevent them from dismantling our chairs, hammering on typewriters, etc.

As I mentioned earlier, there are many symptoms associated with hyperactivity. Clinicians need to be "tuned in" to some of the more subtle symptoms. A

thorough history taking will usually be the best tool for the clinician in determining the presence and etiology of hyperkinesis.

Thorough history taking should also take into account parental history, current family dynamics, etc. Family pathology need not negate "maturational hyperactivity," however clinicians are often not aware of how extremely difficult it may be for the parents and siblings to cope with a hyperactive child. Such a situation can produce great tension, resulting in hostility and/or depression of various family members including the hyperactive child, and acting out behavior including possible child abuse by frustrated parents. Siblings often strike back at the hyperactive child out of anger and jealousy because of the attention the hyperactive child receives. This can occur as open conflict or subtle provocations; the hyperactive child is an easy target. Certainly the reverse can be true, and when great demands are placed on the hyperactive child (e.g. achievement, orderliness, etc.) or when there is a general atmosphere of emotional tension (e.g. marital conflict), the hyperactive child can be greatly worsened with respect to symptoms.

In older children and adolescents as excessive activity declines, increasingly anti-social behavior often develops. Clinicians need to explore for early childhood symptoms and avoid prematurely and perhaps incorrectly diagnosing children and adolescents as anti-social, aggressive reaction, etc.

Psychological testing of hyperactive children prevents some obvious challenges. Although testing may be difficult and not even necessary for establishing a diagnosis (which may be determined through history taking and observation) it is still an important part of the diagnostic and treatment process. The testing needs to focus primarily on intellectual/academic

functioning including the presence of learning deficits, and on current personality status. The former is necessary in order to establish effective educational intervention and the later is necessary in order to determine the degree of psychological damage present as a result of the condition and the conflict it has created with the child's environment. Depression is often present beneath the hostility and anti-social behavior.

I have found that many modifications must be made in testing the hyperactive child. Many tests require several failures on each subtest before concluding it, for example. For the hyperactive child this is usually intolerable and will result in heightened resistance to the evaluation. A relaxed, informal atmosphere is also necessary. Rapport and patience is very important with these children. They seem to respond best, however, with few breaks and firm limits. Encouragement and support is also important. Interpreting tests also requires some flexibility and I find myself using words like "potential," "under optimal conditions," etc. frequently with these children to differentiate between current functioning and potential functioning. I interpret the results for parents and child, though usually seperately so that parents may engage in frank exchanges with me.

Observations of the child, particularly in a play situation is necessary in the diagnostic process. In addition to noting the attention span, the tolerance of frustration for difficult activities, the perceptual-motor coordination, the reponse to limit setting, etc., projective play will help reveal their self concept, mood, and coping strategies.

Treatment/Medications:

The most common form of treatment has been, and continues to be stimulant medications (Harper, Goyette and Conners, 1978). Because of a lack of research

with children under age 6, these medications are generally not administered to children under that age.

Stimulants were first used to treat this disorder in the 1940's (Bender and Coddington, 1942) and they have been increasingly used to the present day. The two most frequently used stimulants are methylphenidate (Ritalin) and dextroamphetamine (Dexedrine). Meeks (1975) reports there has been a gradual shift in preference to Dexedrine, perhaps because it appears to be quicker acting than Ritalin which often requires two to three weeks or more to evaluate. Cylert has been introduced recently. It has been used exclusively for hyperkinesis and its only major difference is that it has a longer half life and is therefore administered less frequently.

The success rates (ie. significant improvement in symptoms) are reported to be 50% to 75% (Meeks, 1975) using stimulant medication. Dosages are generally low initially in order to avoid side effects and are increased gradually to a maximum of 80 mg. of Ritalin or 40 mg. of Dexedrine daily. The drugs take effect rather quickly (15 to 30 minutes) and generally maintain effectiveness for 4 to 6 hours.

There are side effects. The most common are appetite loss, weight loss, and insomnia. Other minor side effects include irritability, depression, stomach pains and headaches. There is some evidence that Ritalin may lower convulsive threshold in patients with a history of seizures (Meeks, 1975). I have observed one case of seizures probably induced by stimulants without a past history of seizures. Toxic psychotic reactions do occur, though rarely. The use of stimulants with hyperactive children who are psychotic or borderline psychotic is generally contra-indicated since these drugs often precipitate a psychotic episode. Minor side effects usually diminish over time or can be corrected by lowering the dosage and gradually increasing it again later.

Sprague and Sleator (1975), have observed the long term effects of stimulant use. They have noted that children on moderate dosages of Ritalin (20 mg./day or more) continuously for 2 or more years will experience a drop in growth rate to 62% of expected weight gain and 75% of expected height gain on average. Some growth rebound occurs after cessation of medication. The drop in growth rate appears to be slightly more pronounced for Dexedrine than Ritalin. However, Ritalin was observed to produce a significant rise in pulse rate particularly in young children, while Dexedrine did not. Many physicians advocate medication holidays in order to minimize the problem and have children off medication during the summer and/or on weekends. These holidays reduce tolerance also, which often occurs with long term use.

Unfortunately, there is often a "rebound phenomenon" (Katz, et.al, 1975) involved with medication holidays. That is, the symptoms of hyperkinesis are dramatically worsened, sometimes even worse than the original symptoms, after cessation of medication for a period of one to several days. It is also common to observe this phenomenon at the end of the day while the child is on medication steadily, since typically medication is prescribed in the morning and early afternoon only initially. In these cases, the teacher will report fine progress, while the parents may report a very negative picture. Such complaints should not be written off then as a product of intolerant or neurotic parents without close investigation.

Sprague and Sleator (1975) also report another problem with stimulant medication. It seems that the optimal dosage range for behavior improvement (the usual target) is higher than the optimal dosage range for cognitive improvement. Consequently, there may have to be a trade off, some hyperkinetic symptoms remaining in order to avoid deleterious effects or cognitive functioning and learning.

Since stimulant medication is not effective with at least 25% of hyperkinetic children, a wide range of other medications have been administered, though stimulant medication remains the first "drug of choice."

Thorazine and Mellaril have been used with children not responsive to stimulants. Although these children often respond with a decline in activity level, rather high dosages are often needed (Katz, et.al, 1975) and there are are no long lasting improvements in social and academic functioning; cognitive functioning in particular seems to be inhibited (Meeks, 1975). There are also common side effects, (weight gain, drowsiness, moodiness, irritability) along with less common side effects, (enuresis, nausea, vomitting, nosebleeds, tremors and jaundice).

Occassionally, tranquilizing medications is combined with stimulants (tranquilizers usually administered at night): to reduce hyperactivity where there are side effects on lower dosages of the stimulant medications; to reduce hyperactivity when the child is less than optimally responsive to stimulant medication alone; to reduce the risk of tolerance; and to reduce the risk of activating psychotic symptoms in borderline or psychotic children.

Tofranil has also been used, though much less commonly. Gittelman-Klein 1975 and Katz, et.al, 1975) report that this tricyclic has been used effectively, particularly with children experiencing seperation anxiety. Again, although activity levels dropped, long term (over 3 months) use resulted in other problems; children became unpleasant and negative and some experienced side effects such as increased appetite, weight gain, drowsiness, dizziness and dry mouth. Sprague and Sleator (1975) have also found that children do not improve in social relatedness or coordination as well as those using Ritalin.

Ritalin and Tofranil have been combined with some children experiencing seperation anxiety. The combination is generally used for a short period of time, a few months, with the Tofranil then gradually withdrawn. Mellaril and Tofranil have also been used (Katz, et.al, 1975) in combination with a hyperactive child who was very sensitive to stimulant medication and did not respond to tranquilizing medication alone. Improvement was noted but "not a completely satisfactory response."

Although the research literature has not discussed these, Atarax (a minor tranquilizer with antihistematic properties) and Benadryl (a sedative-antihistamine) have relieved some symptoms of hyperactivity in clinical practice. Also, particularly in cases of more obvious neurological involvement Dilantin (anticonvulsive) has been effective.

Treatment/Diet:

In 1965 Feingold (1974) observed that elimination of food additives in a woman's diet reduced hives; he also observed that it reduced this woman's rather considerable hostility, which had been the cause for her entering psychiatric treatment some time earlier. In 1966 he found that elimination of food additives in the management of allergies additionally reduced activity level of children who were hyperkinetic. In 1973 at the annual convention of the AMA, Feingold presented his findings on the dietary management of hyperactivity. The controversial treatment has been variously referred to as: the Feingold diet, the Kaiser-Permanente diet, the K-P diet, and the hyperkinesis diet.

There is the assumption that for some susceptible individuals (probably genetically determined) food additives act as toxins on the central nervous system (Weis, 1978). As a toxin, the effects are apparently dose related. That is greater numbers of individuals are affected by increased ingestion of additives and those that are affected are worsened by continued increased ingestion of additives.

Although the Feingold diet has met with enormous skepticism among medical professionals, the lay public has "experimented" with the diet and there are now enough advocates among parents of hyperkinetic children to form numerous Feingold Associations around the country.

Medical reluctance to support this mode of treatment stems in part from the relative lack of research available on the effectiveness of the diet, compared to the extensive research available on stimulant treatment. There have also been concerns about the nutritional value of this diet. However, this later concern has been negated by the research of Harper, et.al (1978) who have found the dietary intake of children on the Feingold diet to meet recommended nutritional standards.

As Weiss (1978) indicated, research on this treatment method is extraordinarily complex because of the difficulties in totally controlling intake, in developing appropriate behavioral measurements,in masking the sureptitious introduction of additives in the double-blind design, and in evaluating the more than 1600 food additives currently used.

The few research studies that have been done to date, have studied only food coloring. Other additives have not been examined. The findings to date have been mixed. Williams et.al (1977), Goyette, et.al (1978) and Conners et. al (1976) have observed significant improvement in behavior using the Feingold diet, but Harley, et.al (1977) did not.

Weiss (1978) found only three of 22 patients on the Feingold diet responding unfavorably to food coloring in a very carefully controlled double-blind experiment. Nevertheless, he indicates that the presence of only one individual adversely affected, establishes the validity of the toxic effect, for <u>some</u> individuals. He also noted that research at the

Hospital for Sick Children in Toranto has found that some hyperkinetic children "challenged" with food coloring experienced deterioration in performance on a cognitive task within an hour of the challenge and this deterioration in performance persisted throughout the day. As the dosage of food coloring was increased, additional children were similarly affected.

Because alleged improvements can be attributed to greater parent-child interaction, or some expectancy for improvement, studies have used individuals on the Feingold diet and have challenged these individuals sureptiously with food coloring. No studies have examined the cumulative effects of many food additives, particularly at "high dosage" levels. Thus, the deleterious effects of the additives might have been more pronounced with the introduction of artificial flavorings and various artificial preservatives in addition to artificial coloring.

Feingold (1974) reports that 50% of the children on the diet respond "fully" and that an additional 25% show enough improvement to stop stimulant medication. He reports that most patients respond to the diet within 10 to 14 days if they are going to improve. Failures to respond he attributes to cheating on the diet (intentionally or not), more gross neurological involvement, or possibly other triggering substances, such as pollutants. Violations in the diet often produce a deterioration in behavior that can appear within one hour and last as long as 5 or 6 days, though typically the effects last only about two days. The amount and duration of the deterioration varies with the amount of additives consumed and the sensitivity of the child. He finds no problems with combining the diet with stimulant medication and he recommends gradual withdrawal of medication after a few weeks.

The Feingold diet in addition to excluding additives, excludes many natural foods which have reportedly produced hyperactive symptoms. These foods to be avoided include, almonds, apples, appricots, most berries, cherries, grapes, peaches, oranges, plums, and prunes. He claims that these foods are chemically related to many of the additives. However, many children on the Feingold diet can be gradually "challenged" to these foods after several weeks on the diet and they can be included in the diet if there are no adverse effects.

Feingold recommends considerable supervision of the child during the first 10 to 14 days. Because hyperactive children tend to be impulsive, they have less ability to control their eating habits during this period. He also recommends keeping a food (including liquids) diary, noting carefully types of food and responses. He feels that the program works best when the whole family attempts the diet, to avoid "singling out" the hyperkinetic child for special attention. Finally, he cautions that allergies can contribute to hyperactivity and block the effectiveness of the diet (milk, wool, pets, feather pillows, etc. may be a problem requiring elimination).

Although there is mounting evidence that this treatment method is helpful for many hyperkinetic children, it is obviously a rigorous program requiring considerable motivation on the part of children and parents. It's effectiveness therefore may be greatest with rather intelligent, well educated families.

Treatment/Psychotherapy:

Most clinicians and psychiatrists currently recommend psychotherapy in addition to medication (Meeks, 1975; Katz et.al, 1975; Douglas, 1975). However, I have observed great numbers of children treated by peditricians,

family physicians, and psychiatrists who have relied solely upon medication. Since success with medication is far from guaranteed (short term or long term) this strategy seems to be rather risky. Furthermore, even in cases where medication is working well, it: does not deal with the residual emotional damage done in past years to both the child and the others in the family; does not prepare the parents in better management skills; perpetrates reliance upon the medication to solve problems; etc. Since it is certainly possible that the medications will not remain effective long term and might well cause undesirable side effects, it would appear prudent to develop alternative treatments while the medication is working well.

The most common form of psychotherapy is with parents, and it is largely educative and supportive in nature, frequently supplemented by behavior modification and/or communication skills interaction. The therapy is often best done in group sessions with several sets of parents facing similar problems. The sessions serve to provide specific information regarding the illness, emotional support, specific management and communication skills, and diminished guilt for having caused or aggravated the condition. This last point is important because parents frequently do "blame themselves" to such a degree that they become unworkable and even rejecting, since they cannot tolerate their own guilt feelings. The popularity of the Feingold Association in many areas of the country may stem at least in part because of the group support achieved at the meetings in addition to the salutary effects of the diet.

As an aside, behavior modification as a primary treatment method is generally only somewhat successful (Katz, et.al, 1975). Changes in behavior are noted, but maintenance of these changes is a problem. The children

often require constant supervision and reinforcement. The method is best used as an ancillary treatment.

Individual psychotherapy appears to be useful with the hyperactive child for many of the same reasons as the parents. The child can acquire information about his/her illness, reduce depression and guilt feelings and improve coping skills. Douglas (1975) advocates specific "training", utilizing role playing and games to verbalize and rehearse strategies to deal with parent conflict, sibling conflict, etc. The therapy can also help this "non-goal-oriented" child to establish goals and priorities. Children often have terrible self images as a result of their constant and often bitter conflicts with those around them. They can benefit from psychotherapy.

Firm limits are generally needed within the therapy session in order for the child to benefit and in order for the therapist to maintain an accepting relationship. This approach is often difficult for therapists who are used to more non-directive approaches.

I have also found behavior modification group therapy to be a useful ancillary treatment. The method lends itself to limit setting and development of appropriate social skills, which hyperkinetic children frequently lack. It is also a method which emphasizes (through positive reinforcement) strengths rather than weaknesses and is therefore generally well received by the children. Group therapy without behavior modification is not advisable unless the behavior has been substantially controlled by medication and/or diet.

Summary:

Hyperkinesis is a very serious and highly complex disorder. It is serious in the sense that it produces long-lasting and profound effects (e.g. low self esteem, poor-cordination, academic deficiencies) which can "outlive" the majority of the symptoms of hyperkinesis. It is complex in the sense that there are many symptoms not all of which are present in each

hyperactive child and in the sense that the treatment methods available are highly varied.

It is important for clinicians to investigate for the possibility of hyperkinesis in virtually all referrals due to the high rate of incidence. This "investigation" need only take the form of observation and brief questions about activity level, academic functioning, coordination, peer relationships, etc. If there are some initial indications of hyperactivity more intensive history taking is necessary. Psychiatric, psychological and neurological evaluations may be useful if this thorough history taking yields positive findings.

The treatment of this disorder requires an eclectic approach. Many forms of medication and psychotherapy are available; dietary management may be appropriate as well, for some children. Clinicians need to maintain a broad, flexible attitude since various treatment approaches are effective with various individuals and since responsiveness to a specific treatment may also change over time, requiring a shift in treatment.

REFERENCES

Bellak, L. Psychiatric Aspects of Minimal Brain Dysfunction in Adults. New York: Grune and Stratton, 1979.

Bender, L. and Coddington, D. The use of amphetamine sulfate in child psychiatry. American Journal of Psychiatry, 1942, 99, 116.

Conners, L. Controlled trial of methylphendate in preschool children with with minimal brain dysfunction. In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Douglas, V. Are drugs enough ? In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Feingold, B. Why Children Are Hyperactive. New York: Random House, 1974.

Goyette, C., Conners, C., Petti, T. and Curtis, L. Effects of artificial colors on hyperkinetic children: a double-blind challenge study. Psychopharmacology Bulletin, 1978, 14, 39.

Gittelman-Klein, R. Pharmacotherapy and Management of pathological seperation anxiety. In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Gittelman-Klein, R. Introduction. In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Guze, S., Wolfgram, E., McKinney, J. and Cantwell, D. Psychiatric illness in the families of convicted criminals. Diseases of the Nervous System, 1967, 28, 651.

Harley, J., Tomasi, L., Ray, R., Eichman, P., Matthews, C., Chun, R., Traisman, E., and Cleeland, C. Hyperactivity and food additives. Paper presented at the annual meeting of the American Psychological Association, San Francisco, 1977.

Harper, P., Goyette, C. and Conners, C. Nutrient intakes of children on the hyperkinesis diet. Journal of the American Dietetic Association, 1978, 73 (5), 515.

Katz, S., Kishore, S., Gittelman-Klein, R. and Klein, D. Clinical pharmacological management of hyperkinetic children. In Gittelman-Klein, R.(ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Klein, D. and Gittelman-Klein, R. Problems in the diagnosis of minimal brain dysfunction and the hyperkinetic syndrome. In Gittelman-Klein, R.(ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Laufer, M. and Denhoff, E. Hyperkinetic behavior syndrome in children. Journal of Pediatrics, 1957, 50, 463.

Meeks, J. Behavior disorders of childhood and adolescence. In Freedman,A., Kaplan, H. and Sadock, B. (eds.) Comprehensive Textbook of Psychiatry, volume 2, Baltimore: Williams and Wilkins Co.,1975, pp. 2127-2136.

Rapaport, J. and Quinn, P. Minor physical anomalies and early developmental deviation: a major biologic subgroup of hyperactive children. In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Ross, D. and Ross, S. Hyperactivity: Research, Theory and Action. New York: Wiley and Sons, 1976.

Safer, D. and Allen, R. Side Effects from long term use of stimulants in children. In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Satterfield, J. and Cantwell, D. Psychopharmacology in the prevention of antisocial and delinquent behavior. In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Sprague, R. and Sleator, E. What is the proper dose of stimulant drugs in children ? In Gittelman-Klein, R. (ed.) Recent Advances in Child Psychopharmacology. New York: Human Sciences Press, 1975.

Weiss, B. Recent research in hyperactivity. Presented at the monthly meeting of the Rochester area Feingold Association, Rochester, NY, November, 1978.

Williams, J., Cram D., Tansig, F. and Webster E. Examining the relative effectiveness of dietary and drug management of hyperkinesis. Paper presented at the annual meeting of Food and Nutrition Liason Commission of the Nutrition Foundation, Palm Springs, January, 1977.